

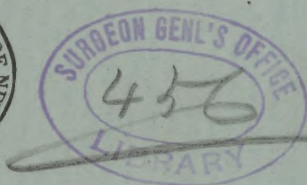
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ON THE ETIOLOGY AND PATHOLOGY
OF TUBERCULOSIS.

BY

HENEAGE GIBBES, M.D.,

PROFESSOR OF PATHOLOGY, UNIVERSITY OF MICHIGAN.



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ON THE ETIOLOGY AND PATHOLOGY OF TUBERCULOSIS.

BY HENEAGE GIBBES, M.D.,

ANN ARBOR, MICHIGAN.

ON examining the lungs of people who have died from disease of these organs, very varied appearances are presented to the naked eye. These range from the small nodules found in acute miliary tuberculosis, to the large ragged cavities found in those cases where the disease has run a more chronic course. There is one feature, however, which is common to all, and that is consolidation. The first step in the disease is the formation of solid masses in the lungs by some abnormal action in the substance of the organ. In this way the affected portions are disabled and cannot perform their functions. The next step is the death of the consolidated part, which then decays and is removed through the air-passages. At the same time the patient undergoes severe wasting and emaciation, the two combined giving us the practical definition of phthisis pulmonalis—viz., progressive consolidation and breaking-down of the lung substance, together with wasting of the body.

So far as the naked-eye appearances go, the disease processes appear to be very much alike, varying only with the duration and severity of the case. When, however, these lungs have been carefully hardened, and sections cut, stained, and mounted, the microscope shows us that they contain two distinct morbid processes—inflammatory and tubercular.

In this classification I include pulmonary phthisis, pulmonary tuberculosis, and acute miliary tuberculosis, leaving out the consolidations produced by acute pneumonia, syphilitic lesions, and hydatid cysts, as they do not come under the heading of this discussion.

Inflammatory or pulmonary phthisis, when examined under the microscope, is found to consist of a mass of broken-down material without any structure, and in which there is no trace of the walls of the air-cells. Everything has become involved in an inflammatory process, which has resulted in the death of the part. The commonest cause producing this condition is broncho-pneumonia, which is essentially lobular in character, from the frequency with which it is

an extension of capillary bronchitis into the substance of the lungs. The first effect of the inflammatory process on the walls of the air-cells is to cause germination of the epithelial cells of which they are composed. The newly-formed cells, together with a varying amount of leucocytes from the capillary bloodvessels, accumulate in the air-cells; these become filled with them, and *then* the capillary vessels become involved, resulting in a mass of inflammatory products through which the circulation is arrested. If the morbid process continues the affected parts lose their vitality, and portions of the lung tissue, varying in size according to the number of air-cells involved, begin to degenerate and undergo caseation, and all the original structure becomes fused into a mass of amorphous *débris*. This is ejected through those air-passages involved in the inflammatory process, and a cavity is formed. Other cavities are formed in the same manner until death takes place. There are conditions, however, in which the destruction of tissue is less or the vitality of the patient greater, and a process of repair takes place, in a cavity by the formation of cicatricial tissue, or in a case where consolidation has not gone on to softening, but remains a solid dead mass; this, acting as an irritant on the surrounding tissues, sets up chronic inflammatory action, and a fibrous capsule is formed round it.

The second form of tuberculosis, although producing a consolidation in the lungs similar to pulmonary phthisis when seen by the naked eye, yet when sections are made of a well-hardened organ the microscope shows the morbid change to be an entirely different one. In tuberculosis there is a new-growth in the lungs. This new-growth consists of a reticular fibroid tissue in which are large cells containing many nuclei—the giant cells. A consolidated mass in a lung the subject of tuberculosis may be as large as that found in pulmonary phthisis, but microscopic examination shows that it is not one large tubercle, but an aggregation of small ones. The reticular tissue in a tubercle is of a peculiar kind—it is distinctly of a fibroid character, and arises from the connective tissue of the lung; but it is not in any way like the adenoid tissue of lymphatic glands, as is sometimes stated. As this fibroid tissue grows, new bloodvessels are formed in it, and by injecting the organ these can be shown to be patent. As this fibroid tissue grows, the nutrition is cut off from the centre of the tubercle, which undergoes a kind of necrosis. In the large consolidations tubercles are found in every stage of development, the older ones with large central necrotic areas, the younger with

only fibroid tissue and one or more giant cells. After a time the central necrosed portion breaks down and a cavity is formed.

In the third form, or acute miliary tuberculosis, we have a disease characterized by its rapid action. Typical cases do not last long before death takes place, *and there are never any tubercle bacilli in the sputum*. Physical signs are also often absent. At the post-mortem examination the lungs are seen to be full of small nodules. When these are examined with the microscope they are found to present two distinct appearances—one form consisting of minute reticular tubercles, the other of small areas of inflammatory breaking-down, without a trace of structure—either newly-formed or of the original lung. *And these two forms never occur together in the same lungs*. This disease must, therefore, be considered, if judged by the lesions found in the lungs, to be of two kinds, and the structure of the lesions shows that the one is inflammatory, the other a new-growth of reticular tubercle. We have, therefore, in phthisis—using the word as a generic term—the following divisions:

1st. Inflammatory, consisting of pneumonic phthisis and the inflammatory form of acute miliary tuberculosis.

2d. Tubercular, consisting of pulmonary tuberculosis and the tubercular form of acute miliary tuberculosis.

The etiology of these different forms must now be considered.

Pneumonic phthisis, whether commencing as broncho-pneumonia or starting *de novo* in the lung substance, as some authorities consider it does, seems to be of as easy explanation as any other inflammatory condition. The inflammatory form of acute miliary tuberculosis also is easily explained, as we know that it often occurs in measles, as does capillary bronchitis. Now, if capillary bronchitis extended from the small bronchioles into the alveoli, we should have a condition of broncho-pneumonia. In a child with deficient expectoration, inflammatory products accumulate in the bronchioles; a sudden fit of coughing would cause these products to be drawn into the lungs in inspiration, they would be distributed into a number of air-cells, and their presence would be sufficient to cause inflammatory action even if they contained no specific morbid products. This would go on until there were a number of foci throughout the lungs which would not all be of the same age, and this is exactly what the microscope reveals to us.

But when we try to explain the relationship of the tubercle bacillus to these two forms of inflammatory disease, we must find

the process in its earliest stage, and this we cannot do in pulmonary phthisis, as the process has gone too far when death takes place. We can, however, in the inflammatory form of acute miliary tuberculosis, find the process at its very first development. Careful examination in a large number of cases has failed to show me tubercle bacilli in this situation, and yet when the process has gone on to caseation they are invariably present in enormous numbers. In pulmonary phthisis they are also present in large numbers, and in those cases of rapid breaking-down called "galloping" consumption, they are also present in enormous numbers, and are noticeable from their size and the number of the so-called spores they contain.

We have, then, the inflammatory condition where the bacillus is absent from the commencing stage, but present when caseation takes place, and we have a full explanation of the whole process without requiring any specific organism.

The etiology of tuberculosis is a more difficult problem, but in this form we can study the earliest stage either in pulmonary tuberculosis or in the tubercular form of acute miliary tuberculosis, as in each tubercles are found in every stage of development. Taking, then, the little tubercle consisting only of a small giant cell with a minute amount of reticular tissue forming round it, we ought to find the bacilli there in the process of organizing this tubercle, but I have never been able to find them. In some cases of acute miliary tuberculosis I have never been able to find a single bacillus after examining hundreds of sections. I have also had cases of pulmonary tuberculosis where the lungs after death were found to be full of cavities, and where no tubercle bacilli could be found in the sputum during life or in the lungs after death. We have, then, cases where the bacilli are absent from the commencement of the lesion, and in some cases absent in the whole course of the disease. In acute miliary tuberculosis, when the bacilli are found, they are not in the necrosed portion as might have been expected, but are found singly in the meshes of the fibroid tissue. They might in this case be considered as the forerunners of the disease; but if they were, they ought also to be found in those tubercles where the disease process is just commencing, and I have never been able to find them there.

Experiments on animals are considered to prove that the tubercle bacillus is the virus of tuberculosis, from the fact that inoculation of an animal will always produce a similar disease, and that the induced lesion is identical with that found in the human lung. I do

not deny that inoculation will produce a lesion in the lungs of a susceptible animal, but I do most emphatically deny that this lesion is similar histologically to that found in miliary tuberculosis. I am now engaged in an investigation into the effects produced on susceptible animals by the inoculation of caseous material from pulmonary tuberculosis and from pulmonary phthisis, and I find, so far, a marked difference, although I have not sufficient data to speak positively. I have found, however, that material from tuberculosis does not at the commencement of the induced lesion cause an inflammatory action, but produces a growth of large cells very similar to those found in scrofula.

The next point to be considered is the production of tuberculosis in animals by the inoculation of a pure cultivation of the tubercle bacillus. Now I would ask, Has it been absolutely proved that any culture of tubercle bacilli is pure—that is, entirely free from any morbid product which was introduced when the material was first taken from the diseased lungs? You are all aware of the careful work done in the case of the jequirity bacillus, where it was thought to be proved beyond the possibility of a doubt that the bacillus was the virus of the disease, and yet it was afterward found that this bacillus had nothing whatever to do with the disease, and the actual poison was isolated from the plant. I think we ought to admit the possibility of a similar condition in the case of the tubercle bacillus. I have for many years held the view that there is some morbid substance of a chemical nature which is intimately associated with the tubercular process, and I think it not improbable that this chemical substance may be carried over with the bacillus in each so-called generation. Soon after my arrival in this country, I found that Dr. Shurly held the same views, and we have always acted on the supposition that the morbid product the cause of tuberculosis is a chemical substance, and have endeavored to neutralize it.

It seems to me, in studying the causation of tuberculosis, that a caseous centre exists somewhere in the body, and that some sudden change in the caseated area results in setting free a morbid substance, which, becoming disseminated, produces a rapid growth of miliary tubercles. This would account for the whole lung becoming studded with tubercles, the morbid products having a peculiar action on the connective tissue of the organ, causing it to increase and form the reticular tissue. The position of the tubercle bacillus is also perfectly consistent with this view, supposing that the origin of the

original caseous centre was infection from bovine tuberculosis. We know that in bovine tuberculosis there are numerous bacilli having the same reaction as those found in the human being, but differing in their size and distribution. These bacilli, having found a suitable pabulum in the caseous centre, increase there. When the centre from any cause becomes broken up, its contents are diffused through the body, and the bacilli with them. This accounts for the presence of a few bacilli in some cases at the margin of a tubercle. If they produced the tubercle, they would surely be found in those that are just commencing.

I think that the bacillus is intimately associated with caseation, as it does not appear on the scene in a growing tubercle until that process has commenced; and in cases of rapid breaking-down of the lung substance, whether in large masses, as in acute pneumonic phthisis, or in small areas, as in disseminated catarrhal pneumonia where there is no tubercular formation whatever, it is invariably present in large numbers, while numerous cases of tuberculosis have been proved to be altogether free from it.

